ACUTE TOXICITY SUMMARY

CARBON DISULFIDE

(carbon bisulfide, carbon sulfide, dithiocarbonic anhydride)

CAS Registry Number: 75-15-0

I. Acute Toxicity Summary (for a 6-hour exposure)

Inhalation reference exposure level	**6,200 μg/m³**
Critical effect(s)	significant reductions in fetal body weight
Hazard Index target(s)	Reproductive/developmental; Nervous System

II. Physical and Chemical Properties (HSDB, 1993 except as noted)

Description	colorless to faintly yellow liquid
Molecular formula	CS_2
Molecular weight	76.14
Density	1.2632 g/cm^3 @ 20° C
Boiling point	46.5°C at 760 mm Hg
Melting point	-11.5°C
Vapor pressure	297 mm Hg @ 20°C
Flashpoint	-30°C (closed cup) (AIHA, 1992)
Explosive limits:	upper = 50% (AIHA, 1992) lower = 1.25%
Solubility	soluble in chloroform, alcohol, ether, benzene, slightly soluble in water
Odor threshold	0.1-0.2 ppm (ACGIH, 1991)
Odor description	Commercially pure CS_2 has a sweetish aromatic odor; industrial grade CS_2 has a rotten cabbage or radish odor (Coppock *et al.*, 1981).

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Metabolites	inorganic sulfates such as thiourea
Conversion factor	1 ppm = 3.11 mg/m³@ 25°C

III. Major Uses or Sources (HSDB, 1993)

The most prominent industrial use of CS_2 is in the production of viscose rayon fibers; it is also used in the production of carbon tetrachloride and cellophane. Carbon disulfide is used as a solvent for rubber, sulfur, oils, resins, and waxes, and has been used for soil fumigation and insect control in stored grain. Industrial processes that produce carbon disulfide as a by-product include coal blast furnaces and oil refining.

IV. Acute Toxicity to Humans

CS_2 is primarily a neurotoxic poison; symptoms indicate both central nervous system (CNS) and peripheral nervous system (PNS) damage. Acute inhalation toxicity, after accidental exposure to very high concentrations, is usually characterized by excitation followed by sulfocarbonic inebriation, similar to drunkenness, and narcosis with extinction of cornea and tendon reflexes (Teisinger, 1971; Bashore and Staley, 1938). Death may occur due to respiratory depression. Recovery from acute exposure may result in motor agitation and disorientation. Other symptoms of acute inhalation toxicity are headache, nausea, garlicky breath, vomiting, dizziness, fatigue, abdominal pain, weak pulse, and palpitations (HSDB, 1993). Hallucinations of sight, smell, hearing, and taste have also been reported following massive vapor exposures. However, many case reports of so-called "acute" poisonings were actually acute exposure and acute onset of symptoms superimposed on chronic inhalation exposure (Gordy and Trumper, 1938). Therefore, it is unknown whether all of the effects described above are due entirely to acute exposure to CS_2. Eye and mucous membrane irritation are also reported as symptoms of acute CS_2 exposure (HSDB, 1993). However, experimental exposure to the pure gas has not resulted in this effect (Beauchamp *et al*., 1983; Du Pont, 1981). It is likely that the irritant effects attributed to CS_2 are due to its combustion products (carbonyl sulfide [COS] and sulfur dioxide [SO_2]) when it burns or to hydrogen sulfide, a known eye and mucous membrane irritant commonly found in workplace air with CS_2 in viscose rayon facilities (Beauchamp *et al*., 1983; Bashore and Staley, 1938; Spyker *et al*., 1982).

Chronic, subchronic, and, in some cases, subacute inhalation exposure to relatively low concentrations of CS_2 have resulted in severe CNS and PNS effects with sequelae different from those seen with acute exposure. The vast majority of the published literature on CS_2 exposure describes long-term or occupational toxicity rather than acute toxicity. Vigliani (1954) reported that viscose rayon workers developed severe CS_2 toxicity from 4-5 hour daily exposures to 1-2 mg/l (322-643 ppm) for as little as 2 months. Symptoms included polyneuritis, psychosis, gastric disturbances, headaches, vertigo, impotence, tremors, sleep disturbances, and myopathy. Concentrations of 0.40 to 0.50 mg/l (129-161 ppm) caused toxicity after 1 or more years of work, while exposure to 0.15-0.20 mg/l (48-64 ppm) did not result in cases of toxicity. Paluch (1948) reported that viscose rayon workers occupationally exposed to 283-370 ppm CS_2 (daily exposure duration unknown) developed serious CNS and PNS effects such as severe headaches, paresthesia of the upper and lower extremities, marked polyneuritis, and neurotic/psychotic behavior. However, one worker exposed to this level of CS_2 for only 8 days experienced severe headaches, psychotic behavior, and optical hallucinations.

Because of improvements in technology and hygienic conditions in viscose rayon factories in developed countries, there have been few, if any, recent reports of acute or chronic toxicity due to occupational exposure from these countries (Teisinger, 1971).

Spyker *et al*. (1982) reported an exposure that occurred following an accidental spill in which a railroad tank car that was leaking CS_2 caught fire. Twenty-seven people, mainly first responders (police and firefighters), were subsequently admitted to a hospital due to exposure. Symptoms

included (in order of frequency) headache, dizziness, nausea, burning of throat, lips, or skin, shortness of breath or chest pain, impotence, and vomiting. No significant changes were observed in FEV_1, FVC, or diffusing capacity and all subjective complaints were transient. However, changes in slow (i.e., not rushed or forced) vital capacity and arterial partial pressure of oxygen were observed, which suggest mild inflammation in small airways. Airborne CS_2 levels of 20 ppm were measured at a nearby undisclosed site during transfer of the chemical to an intact railroad tank car. While the effects reported may have been due to CS_2, it is likely that some or all the effects, particularly the throat, skin, and pulmonary irritation, were due to combustion products such as SO_2 and COS (Spyker *et al*., 1982; Beauchamp *et al*., 1983).

Predisposing Conditions for Carbon Disulfide Toxicity

Medical: Persons with disorders of the central nervous system, eyes, cardiovascular system, kidneys, and liver may be more sensitive to CS_2 (Reprotext, 1999). Persons taking disulfiram (Antabuse) may be more sensitive to CS_2 (Brugnone et al, 1992; Caroldi et al, 1994) since disulfiram is metabolized to CS_2.

Chemical: Human subjects exposed for 6 hours to 10 ppm (30 mg/m³) CS_2 exhibited an inhibition of oxidative N-demethylation (Mack *et al*., 1974). In persons using drugs such as analgesics, hypnotics, antidiabetics, and anticonvulsants, which are metabolized by oxidative N-demethylation, critical elevations in the plasma levels of these agents may be observed following exposure to CS_2. Persons exposed to other neurotoxicants may be at increased risk during carbon disulfide exposure (Reprotext, 1999).

V. Acute Toxicity to Laboratory Animals

Izmerov (1982) reports a 2 hour LC_{50} of 10,000 mg/m³ (3,215 ppm) in mice. Kuljak *et al*. (1974) reports that an "average" lethal concentration (LC_m) of 4,500 ppm over 30 minutes resulted in 17 deaths out of 30 mice. Exposure to 3,000 ppm for 30 minutes/day for 3 days resulted in 21 deaths out of 30 mice. An unpublished report (PPG Industries, 1978) observed a 1-hour LC_{50} of 15,500 ppm in rats. Intraperitoneal injection of 400 mg/kg CS_2 in male guinea pigs resulted in the death of 3 of the 4 test animals within 24 hours (Divincenzo and Krasavage, 1974).

In an unpublished study, exposure of 6 rats to 3,000 ppm CS_2 for 4 hours resulted in no deaths during the exposure or during the 14 day post-exposure observation period (DuPont, 1966). Adverse effects during exposure included tachypnea, ptosis (drooping of eyelids), incoordination, chromodacyorrhea (red fluid emanating from the eyes), and gasping. Weight loss, hyperexcitability, and dyspnea were noted 24 hours post-exposure. Exposure of 6 rats to 3,500 ppm for 4 hours resulted in death of all animals during exposure or before 2 hours post-exposure. Adverse effects similar to the ones previously mentioned were noted, in addition to salivation, aimless wandering, and prostration. Autopsy of 2 rats revealed pleural effusion, dark red and edematous lungs, petechial lung hemorrhages, and pulmonary hyperemia. Changes in other organs were seen but not reported. In another acute inhalation study by the same laboratory,

head-only exposure of rats (4 per group) to 1,660, 8,760, 35,100, or 81,100 ppm CS_2 for 10 minutes did not result in significant respiratory rate depression or overt clinical signs of toxicity (DuPont, 1981). Therefore, CS_2 was not considered by the investigators to be a direct-acting respiratory irritant.

In a range finding portion for a reproductive/developmental toxicity study, 6 pregnant rabbits were exposed to 3,000 ppm CS_2 for 6 hours on day 6 of gestation (PAI, 1991). Four of 6 animals died during exposure and the other 2 were moribund at the end of exposure and were euthanized. No gross lesions were observed but the rabbits exhibited tremors, labored breathing, and apparent anoxia. The 4 animals that died during exposure did not struggle or convulse prior to death. Pregnant rabbits exposed daily (6 hours/day on gestation days 6-18) to 1,000 ppm CS_2 showed only occasional transient signs of toxicity, including ataxia, tremors, and decreased food consumption. Rabbits exposed to 600 ppm or lower showed no signs of exposure-related effects.

Several subchronic studies have reported acute effects in experimental animals soon after initiation of exposure. Wilmarth *et al*. (1993) observed a narcotic-like stupor in rats during 10-hour exposure to 600 or 800 ppm CS_2. Eight-hour exposure of dogs to 404 ppm resulted in drowsiness, stumbling, staggering, and tremors immediately after leaving the exposure chamber (Lewey *et al*., 1941). Exposure of cats to a nominal concentration of 8-10 mg/l (2,560-3,210 ppm) for 2-3 hours resulted in restlessness and excitement early in the exposure; apathy, and occasionally coma, occurred later (Ferraro *et al*., 1941). Other signs of CS_2 toxicity during exposure were salivation, dyspnea, tremors, and muscular jerks. Vomiting was seen occasionally but convulsions were observed in only one instance.

VI. Reproductive or Developmental Toxicity

Carbon disulfide is listed under California Proposition 65 (Cal/EPA, Safe Drinking Water and Toxic Enforcement Act of 1986) as a reproductive hazard with male, female, and developmental endpoints. No teratogenic effects were observed in rats and rabbits exposed to 40 ppm (120 mg/m³) CS_2 for 6 hours per day on days 1-19 or 1-24 of gestation, respectively (Hardin *et al*., 1981). The U.S.EPA NOAEL, from which the Proposition 65 NOAEL for developmental endpoints was adopted, is based on these data (IRIS, 1994).

In a reproductive toxicity study, groups of 15 female rats were exposed to 125, 250, and 500 ppm carbon disulfide 6 hours per day from 14 days prior to mating through day 19 of gestation (CMA, 1993). A concurrent control group of 24 female rats was included in the study. The dams were allowed to deliver normally and both pups and dams were observed through day 21 of lactation. Signs of irritation (clear fluid around the eyes and reddening around the nose) were observed in dams immediately following exposure to 500 ppm. A slight decrease in food consumption was observed between days 15-20 of gestation in dams exposed to 500 ppm. Difficulty with delivery (dystocia) was observed in 2 dams and total litter loss was observed in 3 dams from the 500 ppm group. Increased pup mortality, decreased pup viability, and decreased mean litter size were also observed in this group.

In another study, pregnant rats (17-22 per exposure group; 40 controls) were exposed to 0, 100, 200, 400, or 800 ppm carbon disulfide 6 hours per day on days 6-20 of gestation (Saillenfait *et al*., 1989). A statistically significant reduction in maternal body weight gain was observed in rats exposed to 400 or 800 ppm carbon disulfide. Fetal body weights were also statistically significantly reduced in these exposure groups. A statistically significant increase in the incidence of unossified sternebrae was observed at 800 ppm. An increase in the incidence of club foot at 400 and 800 ppm was not statistically significant.

In a developmental toxicity study conducted by PAI (1991), pregnant rabbits in groups of 24 were exposed to 0, 60, 100, 300, 600, or 1,200 ppm carbon disulfide 6 hours per day on days 6-18 of gestation. In dams exposed to 1,200 ppm, statistically significant decreases in maternal weight gain and clinical signs of toxicity including ataxia, low food consumption, labored respiration, wheezing, tremors, and abortion with bloody excretion involving the death of two animals, were observed. No exposure-related signs of maternal toxicity were observed in does of the other dose groups. In this study, post implantation loss had a significantly higher incidence in does exposed to 600 or 1,200 ppm. Total resorption was observed in 2/22 and 14/21 litters of the 600 ppm and 1,200 ppm exposure groups, respectively. Mean fetal body weight was significantly reduced in the 600 and 1200 ppm exposure groups. In the 1,200 ppm group, the total incidence of skeletal and visceral malformations was significantly increased; however, no single malformation accounted for this increase. In the lower dose groups, significant increases in skeletal malformations were observed in the incidences of rudimentary 13th ribs, extra ribs, extrathoracic vertebrae, or hypoplastic pubis. The malformations in the lower dose groups did not appear to be dose-related and were within the range of historical control data presented by the authors.

In a multigenerational reproductive study, pregnant rats (F_0) inhaled 0.03-200 mg/m³ (0.01-60 ppm) CS_2 for 8 hours per day for the duration of gestation (Tabacova *et al*., 1983). When the healthy pregnant female offspring of the F_0 rats (F_1) were exposed to CS_2 during gestation at levels identical to their prenatal exposure, the progeny of F_1 (F_2) had significantly more malformations than the F_1 generation or the progeny of unexposed rats. For example, exposure at 0.03 mg/m³ was non-teratogenic in the F_1 generation yet had teratogenic effects on the F_2 generation. At the highest level of exposure (200 mg/m³), 38% of the first generation (F_1) exhibited some malformations and 53% of the progeny of this generation (F_2) were malformed while no malformations were observed in controls. The LOAEL for teratogenic effects in the first generation was 100 mg/m³ (30 ppm). Teratogenic endpoints observed in a dose dependent manner within the same generation included gross malformations such as club foot and hypognathia, and CNS abnormalities such as hydrocephalus and microcephalus, in addition to decreased levels of hepatic aniline hydroxylase and aminopyrine N-demethylase. The purity of the CS_2 was not reported, nor was the method of air sampling. It is not clear from the paper if concentrations were measured from the input lines and whether there was potential condensation on fur, cage walls, or food. The study design and the toxicological endpoints observed may be valid, but the dose levels may not have been adequately determined.

Male rats exposed to approximately 610 ppm (1,900 mg/m³) CS_2 for 6 hours per day, 5 days per week for 10 weeks resulted in significant changes in copulatory behavior by the fourth week and reduction in sperm counts by the seventh week (Zenick *et al*., 1984). Caudal epididymal sperm

counts were not depressed and the testes appeared histologically normal. These findings suggest that CS_2 does not exert a direct effect on the testes, but may instead interfere with sperm transport and ejaculation. No significant adverse effects on male rat reproductive parameters were observed following 1 week of exposure to 610 ppm CS_2.

Few reproductive studies exist of human CS_2 exposures. Studies of rayon worker groups suggest that occupational exposure to carbon disulfide may result in reproductive abnormalities. In a cross-sectional study, the rate of spontaneous abortion in women employed in the viscose rayon industry was found to be elevated compared to the rate among women employed in other industrial production, excluding paper products or chemical factories (Hemminki and Niemi, 1982). In this study, women whose husbands worked in the viscose rayon industry also had increased rates of spontaneous abortion. However, this study was exploratory in nature and has yet to be validated.

VII. Derivation of Acute Reference Exposure Level and Other Severity Levels (for a 1-hour exposure)

Mild Adverse Effect Level

Because the most sensitive endpoint found in the literature was developmental toxicity, a potentially disabling effect, there is no mild adverse effect level available for CS_2.

Reference Exposure Level for a 6 hour exposure (protective against severe adverse effects): 2.0 ppm (6,200 μg/m³)

Study	Saillenfait *et al*., 1989
Study population	pregnant rats
Exposure method	inhalation of 0, 100, 200, 400, and 800 ppm on days 6-20 of gestation
Critical effects	significant reductions in fetal body weight
LOAEL	400 ppm
NOAEL	200 ppm
Exposure duration	6 hours
LOAEL uncertainty factor	1
Interspecies uncertainty factor	10
Intraspecies uncertainty factor	10
Cumulative uncertainty factor	100
Reference Exposure Level	2.0 ppm (6.2 mg/m³; 6,200 μg/m³)

Level Protective Against Life-threatening Effects

Animal data suggest that subchronic exposure to 1,000 ppm or less does not result in life-threatening effects while exposure to 3,000 ppm or more for several hours can be lethal. However, the experimental animal lethality studies do not provide enough data for a reliable 1-hour life threatening level. Kuljak *et al*. (1974) reported a 30 minute LC_m of approximately 4,500

ppm in mice. However, since the methods used were relatively primitive and the resulting LC_m does not agree with any other animal exposure data, this study was not considered for life threatening level determination. The studies by DuPont (1966, 1981) observed a steep dose-response for lethality in rats following 4-hour exposure to 3,000 (0% lethality) or 3,500 ppm (100% lethality) CS_2. However, 10 minute exposure of rats to 81,100 ppm did not result in any observable effects. A 1-hour LC_{50} of 15,500 ppm in rats is reported, but no other information is provided (PPG Industries, 1978). In pregnant rabbits, 3,000 ppm for 6 hours produced high mortality while exposure to 1,000 ppm during gestation (6 hours/day) produced little or no effects. Because of the steep dose-response curve for lethality and the lack of lethality data approximating 1 hour of exposure, the life threatening level is based on high-level human occupational exposures to CS_2 that may result in non-lethal effects. These effects were considered comparable to a NOAEL for lethality.

Vigliani (1954) reported that occupational exposure to 322-643 ppm CS_2 4-5 hours/day may result in severe CNS effects such as polyneuritis, psychosis, gastric disturbances, headaches, vertigo, impotence, tremors, sleep disturbances, and myopathy within 2 months. However, no life-threatening effects were reported. Therefore, exposure to 643 ppm for 5 hours, the highest reported human exposure, represents a free-standing NOAEL for life threatening level effects (lethality) in humans. An equivalent 1-hour exposure concentration was estimated from the 5-hour NOAEL using the equation $C^n \times T = K$, where n = 2, resulting in a 1-hour level of 1,438 ppm. An uncertainty factor of 10 was applied to account for sensitive individuals. The resulting level protective against life-threatening effects of 144 ppm (448 mg /m^3) is appropriately health protective, based on occupational studies (Paluch, 1948; Vigliani, 1954; Toyama and Sukurai, 1967) for a 1-hour exposure to CS_2.

VIII. References

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